- 4.7 (1.7, 11) – atomic-bomb survivors less than 15 years old at time of exposure, with a mean thyroid dose from gamma rays of 0.27 Gy (Ron et al., 1995);
- 9.5 (4.1, 19) – atomic-bomb survivors less than 10 years old at time of exposure, with the same mean thyroid dose (Thompson et al., 1994).

These estimates indicate that the risk to children of age less than 10 years at time of exposure is substantially greater than the risk to children of age 10-15 years. For children exposed to *X* rays, the following excess relative risks of thyroid cancer per Gy have been reported (Ron et al., 1995):

- 9.1 (3.6, 29) – newborn children in Rochester, New York, treated for enlarged thymus gland at ages less than 1 year, with a mean thyroid dose from *X* rays of 1.4 Gy;
- 33 (14, 57) – Israeli children treated for ringworm of the scalp at a mean age of 7 years, with a mean thyroid dose from *X* rays of 0.09 Gy;
- 2.5 (0.6, 26) – children in Chicago, Illinois, treated for enlarged tonsils and adenoids at ages 0-15 years (mean age of 4 years), with a mean thyroid dose from *X* rays 0.59 Gy;
- 7.7 (2.1, 29) – pooled analysis of data on childhood exposures at ages 0-15 years, including data on the atomic-bomb survivors (result is dominated by data on childhood exposures to *X* rays at ages less than 10 years).

An RBE for *X* rays can be estimated from these results by assuming that the probability distribution of the estimated risk in each study is lognormal and calculating ratios of the probability distributions for *X* rays to the distributions for gamma rays. Given that the average ages of the children exposed to *X* rays was 7 years or less, the estimated risk in the atomic-bomb survivors of age less than 10 years is used to estimate RBEs. The 95% confidence intervals of the RBEs obtained from the three separate studies of children exposed to *X* rays are (0.3, 4.2), (1.1, 9.2), and (0.06, 3.5), and the 95% confidence interval obtained using the results of the pooled analysis is (0.2, 4.0). If we assume that the biological effectiveness of *X* rays in humans should not be less than that of high-energy gamma rays, based on the calculated effective quality factor in Fig. 1 (ICRU, 1986), these confidence intervals indicate that the RBE for *X* rays in inducing thyroid cancer in children most likely is in the range of about 1-4.

Additional information on the RBE for *X* rays in inducing thyroid cancer can be obtained from a study in prepubescent rats exposed to *X* rays and beta particles emitted in ^{131}I decay (Lee et al., 1982). The biological effectiveness of ^{131}I beta particles, which have an average energy of 182 keV (Kocher, 1981), should be similar to that of high-energy gamma rays (see discussion in section on RBE factors for electrons). The following central estimates and 95% confidence intervals (in parentheses) of ratios of thyroid tumor incidence from *X* rays to that from ^{131}I beta particles at different mean thyroid doses were obtained: 1.1 (0.32, 3.7) at 0.8 Gy, 1.2 (0.43, 3.2) at 3.3 Gy, and 1.4 (0.24, 7.6) at 8.5 Gy. If the three confidence intervals are averaged, the result is a central estimate (50th percentile) and 95% confidence interval of 1.4 (0.6, 3.6). Thus, although the uncertainties are large and an RBE as high as about 4 cannot be ruled out, the biological effectiveness of *X* rays and ^{131}I beta particles in inducing thyroid cancer in the study animals was about the same, on average.

Finally, we examined results obtained from epidemiological studies of cancers at other sites, including the colon, lung, skin, female breast, and bladder (UNSCEAR, 2000). The central estimate of the excess relative risk per Gy in populations exposed to *X* rays often was comparable to or less than the central estimate in a similar age group in the atomic-bomb survivors, although some of the lower risks from *X* rays may be influenced by the much higher doses of *X* rays compared with the doses of gamma rays in the atomic-bomb survivors. In those few cases where a higher risk was observed in populations exposed to *X* rays, the difference was less than a factor of 2. In all cases, however, uncertainties in the risk estimates are sufficiently large that an RBE for *X* rays substantially greater than 1 cannot be ruled out.

The results of epidemiological studies described above lead to the following observations. First, there is no evident difference in the effectiveness of *X* rays in inducing thyroid cancers compared with cancers at other sites. Second, the uncertainties in the results of epidemiological studies are sufficiently large that an upper confidence limit of the RBE factor as high as 5.0, as we have assumed based on radiobiological studies, cannot be ruled out. Third, although uncertainties in the results of epidemiological studies are large, in no cases is a central estimate of an RBE for *X* rays as high as 4 obtained. Based on considerations of statistical uncertainties alone, an occasional high estimate of RBE would be expected. Finally, the epidemiological data do not rule out an assumption that the biological effectiveness *X* rays in inducing cancers in humans is the same as that of high-energy gamma rays.

Based on the evidence obtained from all the radiobiological and epidemiological studies discussed above, we describe the RBE factor for orthovoltage *X* rays and other lower-energy photons, $\overline{\mathrm{RBE}}_{\mathrm{M}}$, to be used in estimating cancer risks at low doses and low dose rates in accordance with eq. (2) by a probability distribution in which a weight of 0.25 is assigned to the value 1.0 and a weight of 0.75 is assigned to a lognormal distribution having a 95% confidence interval between 1.0 and 5.0. That is, we use the results of epidemiological studies to modify the lognormal probability distribution that was based on the results of radiobiological studies by assigning a substantial weight to an assumption that *X* rays and other lower-energy photons have the same biological effectiveness in humans as high-energy gamma rays. The resulting probability distribution of the RBE factor has a 95% confidence interval between 1.0 and 4.7. The 50$^{\mathrm{th}}$ percentile of this distribution is 1.9, and the arithmetic mean is 2.1.

Energy-Dependence of RBE Factor

Based on a calculation of the effective quality factor vs. photon energy given in Fig. 1 (ICRU, 1986), we assume that the RBE factor for orthovoltage *X* rays and other lower-energy photons described above applies at energies of 30-250 keV; the effective quality factor is essentially independent of energy over much of this range. We also note that the effective quality factor at these energies is slightly more than twice the value at the energies of ^{60}Co gamma rays (1.2 and 1.3 MeV); by our reading of the curve in Fig. 1, the difference is a factor of 2.3. This result is in good agreement with the central estimate of the RBE factor described above and, thus, provides support for our assumption.

An assumption that the RBE factor applies at photon energies as low as 30 keV is supported by calculations of the biological effectiveness of 60- and 80-kVp *X* rays relative to gamma rays from the Hiroshima and Nagasaki atomic bombs for a number of specific endpoints, including chromosomal aberrations in human lymphocytes, induction of mutations in human fibroblasts, and oncongenic transformation in C3H10T½ mouse cells (Brenner, 1999). RBEs at low doses between 1.6 and 2.0 were calculated. The differences between these values and the value of 2.3 inferred from the calculation in Fig. 1 are due, in part, to differences in the assumed responses as a function of lineal energy and to an assumption that the average energies of gamma rays from the atomic bombs were somewhat less than the energies of ^{60}Co gamma rays. The biological effectiveness of photons of energy less than 30 keV is considered below.

The calculation of the effective quality factor shown in Fig. 1 indicates that the biological effectiveness increases as the photon energy decreases below 30 keV. For example, using the calculation in Fig. 1, Brenner and Amols (1989) estimated that 23 kVp *X* rays should be approximately 1.3 times more effective than 44-250 kVp *X* rays in inducing breast cancer. Thus, based on the calculation in Fig. 1, we assume that the probability distribution of the RBE factor for photons of energy 30-250 keV should be increased when the energy is less than 30 keV, and we represent this increase by a factor which is described by a triangular probability distribution having a lower bound of 1.0, a mode of 1.3, and an upper bound of 1.6.

Summary

Cancer risks in humans from exposure to photons are estimated using an approach represented by eq. (2). Specifically, the risk per unit absorbed dose from exposure to photons (γ) at low doses and low dose rates is estimated as

$$R_\gamma = \overline{\mathrm{RBE}}_{\gamma,\mathrm{M}} \times \mathrm{AF}_\gamma \times \frac{R_{\gamma,\mathrm{H}}}{\mathrm{DDREF}_\gamma}, \qquad (7)$$

where $\overline{\mathrm{RBE}}_{\gamma,\mathrm{M}}$ is the RBE factor for photons of energy 30-250 keV at low doses and low dose rates, AF_γ is an adjustment factor that represents an increase in biological effectiveness when the photon energy is less than 30 keV, and $R_{\gamma,\mathrm{H}}$ and DDREF_γ are the same as in eq. (2). The relative biological effectiveness of all photons of energy greater than 250 keV is assumed to be unity.

Given the assumed probability distributions of $\overline{\mathrm{RBE}}_{\gamma,\mathrm{M}}$ at energies of 30-250 keV and the adjustment factor, AF_γ, at energies less than 30 keV, a small probability is assigned to an RBE factor less than 1.0 at energies less than 250 keV. In all such cases, however, the lower tail of the probability distribution of the RBE factor should be truncated at 1.0. This truncation is based on the calculated effective quality factor in Fig. 1, which indicates that the biological effectiveness of lower-energy photons should not be less than that of high-energy gamma rays.

Acute exposure to photons also is of concern in exposures of workers and the public. Cancer risks in humans from acute exposure to photons also are estimated using eq. (7), and the two terms describing the biological effectiveness of photons of energy less than 250 keV relative to high acute doses of high-energy gamma rays are the same. However, the dose and dose-rate effectiveness factor ($DDREF_\gamma$) is different, and generally lower, than in cases of chronic exposure. The probability distribution of $DDREF_\gamma$ for acute exposure is assumed to depend on the magnitude of the dose, and a single value of 1.0 is assumed at absorbed doses above 0.2 Gy (see footnote a in Table 14). Thus, the biological effectiveness of lower-energy photons relative to high-energy gamma rays is assumed to be independent of dose and dose rate under similar conditions of exposure to the two radiations.

RBE FACTORS FOR ELECTRONS

With the exception of the low-energy electrons emitted in beta decay of ^3H, there have been few studies of the biological effectiveness of electrons relative to gamma rays or *X* rays. In this section, we first develop a probability distribution of the RBE factor for beta particles emitted in ^3H decay. The spectrum of electrons in ^3H decay has an average energy of 5.7 keV and a maximum energy of 18.6 keV (Kocher, 1981). We then consider the biological effectiveness of other electrons, including low-energy Auger electrons.

RBE Factor for Tritium Beta Particles

Many studies have shown that beta particles emitted in ^3H decay are biologically more effective than gamma rays in inducing stochastic effects (NCRP, 1990; Straume and Carsten, 1993). Estimates of RBE obtained from studies reviewed by Straume and Carsten (1993), including studies in which the reference radiation was *X* rays, are summarized in Tables 10-13.

For purposes of estimating an RBE factor for ^3H beta particles that is consistent with the RBE factors developed for the other radiation types, the relevant studies are those in which the reference radiation was gamma rays. In most studies using gamma rays, the reference radiation was delivered chronically to match the conditions of exposure to ^3H beta particles. Thus, cancer risks in humans per unit dose of ^3H beta particles are estimated using the approach in eq. (2), which applies at low doses and dose rates. If we assume that the DDREF for ^3H beta particles in the various studies is about the same as the DDREF for the reference radiation, RBEs obtained under conditions of chronic exposure in Tables 10-13 provide estimates of RBE_M.

Based on the RBEs for chronic or sub-acute exposure to gamma rays in Tables 10-13, we describe the RBE factor for ^3H beta particles at low doses and low dose rates, $\overline{RBE}_M$, by a lognormal probability distribution having a 95% confidence interval between 1.2 and 6.0. This distribution has a geometric mean and geometric standard deviation of 2.7 and 1.5, respectively, and an arithmetic mean of 2.9. Given the residence half-time of tritiated water in soft tissues of

about 10 days (ICRP, 1979) and the much longer half-life of ^{3}H (12.3 years), acute exposure to beta particles emitted by ^{3}H is not expected to be of concern.

In a previous analysis by *SENES* Oak Ridge (Thomas and Hoffman, 2000), the RBE factor for ^{3}H beta particles was described by a triangular probability distribution having a lower bound of 1.0, a mode of 2.0, and an upper bound of 5.0. The lognormal probability distribution described above is similar to the previous assumption. However, based on the data summarized in Tables 10-13, an RBE greater than 5 cannot be ruled out. The upper tail of the lognormal probability distribution represents an assumption that the RBE factor could be 5 or greater, and that a reasonable upper bound cannot be determined with certainty. The substantial probability assigned to an RBE factor of 4 or greater (about 15%) also is intended to take into account that the RBE for organically-bound tritium appears to be 2-3 times higher than the RBE for HTO or ^{3}H incorporated into amino acids (see Tables 11 and 13). This is an important consideration when some HTO taken into the body becomes organically-bound before it is excreted (Straume and Carsten, 1993).

The assumed probability distribution of the RBE factor for ^{3}H beta particles is nearly the same as the probability distribution for photons of energy less than 30 keV discussed in the previous section. This consistency is expected when, as discussed below, the energies of electrons that deliver an absorbed dose are similar. The assumed probability distribution also is supported by the data in Tables 10-13 which indicate that biological effectiveness of ^{3}H beta particles is about the same as that of *X* rays.

RBE Factor for Other Electrons

Since the energies of ^{3}H beta particles are very low, we considered whether other electrons, especially those of higher energy, should be assigned an RBE factor greater than unity. In radiation protection, all such electrons generally are assumed to have the same biological effectiveness as high-energy gamma rays (see Table 1). The study of the biological effectiveness of *X* rays and beta particles from ^{131}I decay in inducing thyroid cancer in rats by Lee et al. (1982) discussed previously is the only study we are aware of that was specifically designed to investigate the biological effectiveness of higher-energy electrons. In the absence of extensive radiobiological data, we address this question using the following arguments.

In the previous section, data on the biological effectiveness of *X* rays and a calculation of the effective quality factor vs. photon energy shown in Fig. 1 were used to develop RBE factors greater than 1.0 for photons of energy less than 250 keV. Since the absorbed dose from irradiation by photons is due almost entirely to energetic secondary electrons produced by interactions of the photons in tissue, information on the biological effectiveness of photons can be used to infer the biological effectiveness of electrons. That is, an RBE factor for photons of a given energy essentially describes the biological effectiveness of the secondary electrons produced by the first interactions of these photons in tissue.

The energies of secondary electrons produced by interactions of photons in tissue generally decrease with decreasing photon energy. Therefore, electrons produced by interactions of 250-keV photons in tissue are at the highest energies for which the biological effectiveness should be the same as that of lower-energy photons. In tissue, which has an average atomic number of 7 (Shleien et al., 1998), Compton scattering is the dominant interaction at a photon energy of 250 keV [see Fig. A.1 of NCRP (1991) and Figs. 5.1 and 5.2 of Shleien et al. (1998)]. At this energy, the spectrum of secondary electrons produced by Compton scattering has a maximum energy of 124 keV and an average energy of 60 keV (Turner, 1995). In contrast, the energy of secondary electrons produced by the photoelectric effect in tissue at this energy is nearly 250 keV, since the binding energies of electrons in atoms of the elements comprising tissue are about 3 keV or less (Shleien et al., 1998). At 250 keV, however, photoelectrons are produced in only about 0.1% of all interactions [see Fig. 5.2 of Shleien et al. (1988)] and, thus, have little effect on the average energy of secondary electrons.

As the incident photon energy decreases below 250 keV, the photoelectric effect increases in importance relative to Compton scattering, and becomes the dominant interaction in tissue at energies less than about 30 keV (Schleien et al., 1998; NCRP, 1991). At this energy, the average and maximum energies of secondary electrons in Compton scattering are about 1.5 and 3 keV, respectively, and the average energy of photoelectrons is nearly 30 keV. Thus, the average energy of secondary electrons at this photon energy is about 15 keV. This result is of interest because we have assumed, based on the calculated effective quality factor shown in Fig. 1, that the biological effectiveness of photons of energy less than 30 keV is higher than at 30-250 keV. Thus, the same increase should apply to electrons of energy less than about 15 keV. As the photon energy decreases below 30 keV, the energies of secondary electrons produced in tissue approach the incident photon energy, due to the increasing dominance of the photoelectric effect and the low binding energies of atomic electrons in tissue. At a photon energy of 20 keV, for example, the energies of secondary electrons are little different from the photon energy.

We believe that three conclusions can be drawn from this analysis. First, at electron energies greater than 60 keV, the RBE factor can be assumed to be unity, without uncertainty, to conform to the assumption for photons of energy greater than 250 keV. Second, at electron energies in the range of 15-60 keV, the RBE factor can be assumed to be the same as the RBE factor for photons of energy 30-250 keV. Third, at electron energies less than 15 keV, but possibly excluding low-energy Auger electrons as discussed below, the RBE factor can be assumed to be the same as the RBE factor for photons of energy less than 30 keV.

At electron energies less than 15 keV, however, we believe it is preferable to assume that the RBE factor is the same as the RBE factor for beta particles emitted in ^{3}H decay. As noted above, the RBE factors for ^{3}H beta particles and photons of energy less than 30 keV are nearly the same. In our view, an advantage of using the RBE factor for ^{3}H beta particles is that it is based directly on radiobiological data. We also note that the energies in the spectrum of ^{3}H beta particles span the energies over which the RBE factor at the lowest electron energies is applied.

The appropriate RBE factor at electron energies of 15-60 keV or less than 15 keV should be applied to beta-emitting radionuclides when the average energy of the continuous spectrum of beta particles is less than 60 keV. Use of the average energy of beta particles is reasonable when the argument to assume an RBE factor greater than 1.0 at energies less than 60 keV is based on the average energy of the continuous spectrum of secondary electrons in Compton scattering. The appropriate RBE factor also should be applied to discrete internal conversion electrons of energy less than 60 keV emitted by radionuclides.[17] In these cases, however, an RBE factor needs to be taken into account only when the average energy of low-energy internal conversion electrons per decay of a radionuclide is significant compared with the average energies per decay of other radiations having a short range in tissue, including internal conversion electrons of energy greater than 60 keV, beta particles, and alpha particles. Application of the RBE factors to Auger electrons is discussed below.

RBE Factors for Auger Electrons

Radionuclides that emit Auger electrons[18] require special consideration, due to the very low energies of these radiations (often a few keV or less) and their short range in matter (less than 0.1 μm). The ICRP (1991) and the NCRP (1993) recommend that Auger electrons emitted by radionuclides that are incorporated into DNA should not be assigned a radiation weighting factor of 1 (see Table 1), since it is unreasonable to average the absorbed dose over the whole mass of DNA. Techniques of microdosimetry are considered more appropriate in such cases.

Limited data on the biological effectiveness of Auger electrons are summarized by the ICRP (1991). When Auger emitters penetrate a cell but are not incorporated into DNA, RBEs for a number of endpoints, including cell killing, are in the range of 1.5-8. Such RBEs are similar to values for low-energy beta particles from ^{3}H decay discussed previously. However, when Auger emitters, such as ^{125}I, are incorporated into DNA, RBEs in the range of 20-40 have been found for such endpoints as cell transformation. Such high RBEs are supported by calculated patterns of energy deposition.

When information on whether an Auger-emitting radionuclide is incorporated into DNA of an exposed individual is lacking, we believe that Auger electrons should be treated in the same

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[17]Internal conversion is the process by which the energy difference between an initial and final state in an atomic nucleus is transferred directly to a bound atomic electron, which is then ejected from the atom. The emission of internal conversion electrons competes with the emission of gamma rays, and it increases in importance as the atomic number increases.

[18]The emission of Auger electrons competes with the emission of *X* rays as a means of carrying off the energy released when a vacant energy state of electrons in an atom is filled by an electron in a higher energy state. In the Auger process, the filling of the vacant energy state is accompanied by the simultaneous ejection of an electron in a higher energy state from the atom. Auger electrons usually are important only when a radionuclide decays by electron capture or an isomeric transition (Kocher, 1981).

way as other low-energy electrons. Thus, for example, when the energy of Auger electrons is less than 15 keV, the RBE factor that applies to low-energy beta particles emitted in ^{3}H decay should be used. When Auger electrons are important compared with other low-energy electrons, their energies are nearly always less than 15 keV (Kocher, 1981).

When an Auger-emitting radionuclide is known to be incorporated into DNA, however, we do not believe that a credible probability distribution of the RBE factor can be developed based on available information. Although the RBE factor in such cases should be substantially higher than the RBE factor that applies to ^{3}H beta particles, there are potentially important uncertainties including, for example, the fraction of the activity that is incorporated into DNA, the dependence of RBE on the energy of Auger electrons, and the dependence of RBE on dose when cell killing could occur. Thus, we support the recommendation of the ICRP (1991) and the NCRP (1993) that the biological effectiveness of Auger emitters that are incorporated into DNA should be handled as special cases using techniques of microdosimetry.

Summary

Cancer risks in humans from exposure to electrons are estimated using an approach represented by eq. (2). Specifically, the risk per unit absorbed dose of electrons (e) at low doses and low dose rates is estimated as

$$R_e = \overline{\mathrm{RBE}}_{e,\mathrm{M}} \times \frac{R_{\gamma,\mathrm{H}}}{\mathrm{DDREF}_\gamma}, \tag{8}$$

where $\overline{\mathrm{RBE}}_{e,\mathrm{M}}$ is the RBE factor for electrons at low doses and low dose rates, and $R_{\gamma,\mathrm{H}}$ and DDREF_γ are the same as in eq. (2). Two RBE factors are specified. The first applies to electrons of energy 15-60 keV, including spectra of beta particles for which the average energy lies in this range. At these energies, the RBE factor is assumed to be the same as the RBE factor for photons of energy 30-250 keV, based on considerations of the energies of secondary electrons produced by interactions of these photons in tissue. At electron energies less than 15 keV, including spectra of beta particles of the appropriate average energy, an RBE factor obtained from radiobiological data on beta particles emitted in decay of ^{3}H is applied, except when an Auger-emitting radionuclide is known to be incorporated into DNA. The relative biological effectiveness of all electrons of energy greater than 60 keV is assumed to be unity. This assumption applies, for example, to the spectrum of beta particles emitted in ^{131}I decay.

Given the assumed probability distributions of $\overline{\mathrm{RBE}}_{e,\mathrm{M}}$ at energies of 60 keV or less, a small probability is assigned to an RBE factor less than 1.0. In all cases, however, the lower tail of the probability distribution of the RBE factor should be truncated at 1.0. This truncation is based on an assumption that the biological effectiveness of low-energy electrons should not be less than that of high-energy gamma rays, and it is consistent with the truncation of probability distributions of RBE factors for lower-energy photons discussed previously.

The assumed RBE factors for electrons would be important in calculating cancer risks and probability of causation whenever intakes of radionuclides that emit low-energy beta particles, internal conversion electrons, or Auger electrons contribute significantly to estimated doses to an organ or tissue of concern. Examples of potentially important radionuclides that emit electrons in the energy range of 15-60 keV include ^{14}C, ^{63}Ni, ^{93}Zr and its decay product ^{93m}Nb, ^{95}Nb, ^{129}I, ^{132}Te, and ^{151}Sm (Kocher, 1981). At energies less than 15 keV, potentially important radionuclides include, in addition to ^{3}H, the beta-emitting radionuclides ^{106}Ru and ^{107}Pd and the Auger-emitting radionuclides ^{51}Cr, ^{55}Fe, ^{57}Co, ^{58}Co, ^{65}Zn, and ^{125}I (Kocher, 1981).

If acute exposure to electrons is of concern, the dose and dose-rate effectiveness factor ($DDREF_\gamma$) is estimated as in cases of acute exposure to photons discussed following eq. (7). Acute exposure to electrons could be important in cases of external exposure, but is unlikely to be important in cases of internal exposure to radionuclides.

SUMMARY OF RBE FACTORS FOR DIFFERENT RADIATIONS

Based on evaluations of information on the biological effectiveness of various types of ionizing radiation, we have developed relative biological effectiveness (RBE) factors for use in calculating the probability of causation of specific cancers in humans. These RBE factors are applied to estimates of cancer risks per unit dose at high doses and high dose rates of high-energy gamma rays, which are obtained mainly from studies in the Japanese atomic-bomb survivors.

The RBE factors developed in this report are expressed as probability distributions. These distributions are intended to represent the current state of knowledge (i.e., uncertainties) in the relevant radiobiological data and any other judgments involved in evaluating the available information. The RBE factors for the different radiations considered in this report are summarized as follows.

Neutrons

Cancer risks per unit absorbed dose at any dose and dose rate of neutrons are estimated using eq. (5). At energies in the range of 0.1-2 MeV, including fission neutrons, the RBE factor at high doses and high dose rates of the reference gamma radiation, $\overline{RBE}_{n,H}$, is described by a lognormal probability distribution having a 95% confidence interval between 1.5 and 30. This distribution has a geometric mean of 6.7 and a geometric standard deviation of 2.2.

At energies outside the range of 0.1-2 MeV, the RBE factor is obtained by scaling (reduction) of the probability distribution of $\overline{RBE}_{n,H}$ by an adjustment factor which is based mainly on the energy dependence of the radiation weighting factor recommended by the ICRP (1991). At energies of 10-100 keV or 2-20 MeV, this adjustment factor is described by a lognormal probability distribution having a 95% confidence interval between 1.0 and 4.0; at energies of <10 keV or >20 MeV, this adjustment factor is described by a lognormal probability

distribution having a 95% confidence interval between 2.0 and 8.0. These distributions have a geometric mean of 2.0 and 4.0, respectively, and a geometric standard deviation of 1.4.

Under conditions of chronic exposure only, a small enhancement factor representing a possible inverse dose-rate effect is applied to the RBE factor for neutrons of any energy. This enhancement factor is described by a discrete probability distribution having 50% of the values at 1.0, 30% at 1.5, 15% at 2.0, and 5% at 3.0.

After all relevant adjustments for the exposure situation of concern are applied to the RBE factor for fission neutrons, $\overline{\text{RBE}}_{n,\text{H}}$, the lower tail of the resulting probability distribution should be truncated at 1.0. This truncation is based on an assumption that the biological effectiveness of neutrons should not be less than that of high-energy gamma rays.

Alpha Particles

Except in cases of exposure to radon and its short-lived decay products, cancer risks per unit absorbed dose at low doses and low dose rates of alpha particles are estimated using eq. (6). The RBE factor at low doses and low dose rates of the reference gamma radiation, $\overline{\text{RBE}}_{\alpha,\text{M}}$, is described by a stepwise-uniform probability distribution having 15% of the values in the range of 1.0-10, 25% in the range of 10-20, 30% in the range of 20-30, 20% in the range of 30-40, 7.5% in the range of 40-60, and 2.5% in the range of 60-100. A small enhancement factor representing a possible inverse dose-rate effect is applied to all exposures to alpha particles emitted by radionuclides. This enhancement factor is described by a discrete probability distribution having 70% of the values at 1.0, 20% at 1.5, 7.5% at 2.0, and 2.5% at 3.0. Since the two probability distributions that apply to alpha particles have a lower bound of 1.0, truncation of the aggregate probability distribution at 1.0, based on an assumption that the biological effectiveness of alpha particles should not be less than that of high-energy gamma rays, is not needed. Acute exposures to alpha particles are not expected to be of concern for workers or the public.

Photons

Cancer risks per unit absorbed dose at low doses and low dose rates of photons are estimated using eq. (7). At energies greater than 250 keV, the RBE factor is assumed to be 1.0, without uncertainty. At energies of 30-250 keV, the RBE factor at low doses and low dose rates of the reference gamma radiation, $\overline{\text{RBE}}_{\gamma,\text{M}}$, is described by a probability distribution in which a weight of 0.25 is assigned to the value 1.0 and a weight of 0.75 is assigned to a lognormal distribution having a 95% confidence interval between 1.0 and 5.0. At energies less than 30 keV, the probability distribution of $\overline{\text{RBE}}_{\gamma,\text{M}}$ is increased by a small adjustment factor which is based on a calculation of the effective quality factor vs. energy by the ICRU (1986). This adjustment factor is described by a triangular probability distribution having a minimum of 1.0, a mode of 1.3, and a maximum of 1.6. The lower tail of the probability distribution of the RBE factor at any energy should be truncated at 1.0, based on an assumption that the biological effectiveness of low-energy photons should not be less than that of high-energy gamma rays.

Cancer risks from acute exposure to photons also are estimated using eq. (7). For acute exposure, however, the dose and dose-rate effectiveness factor, $DDREF_\gamma$, is assigned the single value 1.0 at doses above 0.2 Gy or, at lower doses, a probability distribution that approaches the probability distribution of $DDREF_\gamma$ for chronic exposure as the dose approaches zero.

Electrons

Cancer risks per unit absorbed dose at low doses and low dose rates of electrons are estimated using eq. (8). At energies greater than 60 keV, the RBE factor is assumed to be 1.0, without uncertainty. At energies of 15-60 keV, the RBE factor at low doses and low dose rates of the reference gamma radiation, $\overline{RBE}_{e,M}$, is described by a probability distribution which is the same as the probability distribution of the RBE factor for photons of energy 30-250 keV. At energies less than 15 keV, including beta particles emitted in ^3H decay and low-energy Auger electrons, the RBE factor at low doses and low dose rates is described by a lognormal probability distribution having a 95% confidence interval between 1.2 and 6.0, except Auger-emitting radionuclides that are known to be incorporated into DNA should be handled as special cases using techniques of microdosimetry. The probability distribution at the lowest energies has a geometric mean of 2.7 and a geometric standard deviation of 1.5. The lower tail of the probability distribution of the RBE factor at any energy should be truncated at 1.0, based on an assumption that the biological effectiveness of low-energy electrons should not be less than that of high-energy gamma rays.

Cancer risks from acute exposure to electrons also would be estimated using eq. (8), and the approach to estimating $DDREF_\gamma$ for acute exposure to photons described above would be used. However, acute exposure to electrons is unlikely to be important in exposures of workers and the public.

Summary Table

The probability distributions of RBE factors for the different radiation types developed in this report are summarized in Table 14. For each radiation type and energy, the probability distribution of the RBE factor and any modifying factors is described, and the 95% confidence intervals and central estimates (50^{th} percentiles) are given. For example, the probability distribution of the RBE factor for chronic exposure to neutrons of energy 10-100 keV is obtained by combining the separate probability distributions of the RBE factor for acute exposure to fission neutrons, an adjustment factor to account for the biological effectiveness of 10-100 keV neutrons compared with fission neutrons, denoted by AF_2, and an enhancement factor to account for the inverse dose-rate effect under conditions of chronic exposure, denoted by EF_n.

Table 1. Values of effective quality factor, $\overline{Q}$, and radiation weighting factor, w_R, for selected radiation types currently recommended for use in radiation protection[a]

Radiation type	Effective quality factor[b] ($\overline{Q}$)	Radiation weighting factor[c] (w_R)
Photons		
All energies		1
> 30 keV[d]	1	
Electrons		
All energies[e]		1
> 30 keV	1	
Tritium beta particles	2	
Neutrons		
Unknown energy[f]	25	
< 10 keV		5
10-100 keV		10
100 keV-2 MeV		20
2-20 MeV		10
> 20 MeV		5
Alpha particles	25	20

[a]Distinction between effective quality factor and radiation weighting factor is described in footnote 7 of main text.

[b]Values recommended by ICRU (1986) are based on calculations of quality factor vs. lineal energy in a 1-μm diameter sphere of tissue-equivalent material.

[c]Values recommended by ICRP (1991) and NCRP (1993).

[d]At photon energies less than 30 keV, calculated effective quality factor increases with decreasing energy (see Fig. 1).

[e]Auger electrons emitted in decay of radionuclides incorporated into DNA are excluded [see paragraphs A13 and B67 of ICRP (1991)].

[f]When neutron energy at location of interest in tissue is known, calculated values of $\overline{Q}$ vs. energy shown in Fig. 2 can be used.

Table 2. Summary of values of RBE_M for fission neutrons relative to high-energy gamma rays estimated by expert groups[a]

Biological response	ICRU (1986)	NCRP (1990)
Tumor induction	~3 - ~200	16-59
Life shortening	15-45	10-46
Transformation	35-70	3-80[b]
Cytogenetic studies[c]	40-50	34-53
Genetic endpoints[d]	10-45	5-70[e]

[a]Values of RBE_M apply at the low doses and low dose rates, and are obtained by extrapolation of data on dose-response for neutrons and the reference radiation at higher doses and dose rates; derived values of RBE_M generally are greater than the corresponding values of RBE at higher doses and dose rates. Only values for stochastic endpoints obtained from studies in mammalian systems are given.

[b]Value of 80 was derived from one set of experiments only.

[c]Studies on human lymphocytes in culture.

[d]Studies in mammalian systems only; range of values for genetic endpoints in plant systems estimated by NCRP (1990) is 2-100.

[e]Value of 70 derived from data on specific locus mutations in mice is not necessarily an RBE_M.

Table 3. Values of RBE_H and RBE_M of fission neutrons for life-shortening in various strains of mice derived from analysis of selected studies by Edwards (1999)[a]

Strain and Reference	RBE_H			RBE_M		
	LL[b]	Mean	UL[b]	LL[b]	Mean	UL[b]
RF/Un, Upton et al. (1967)[c]						
Female	2.5	3.5	4.5	2 5	3.5 15	4.5 55
RFM, Storer et al. (1979)						
Female	2.1	2.4	2.7	6.7	7.5	8.3
BALB/c, Storer and Ullrich (1983)						
Female	3.5	4.5	5.5	12	15	18
B6CF1, Carnes et al. (1989)						
Male	7	8	9	35	50	65
Female	8.5	9.5	10.5	34	45	56

[a]See Table 3 of Edwards (1999). RBE_H is RBE at high acute doses of reference high-energy gamma radiation, and RBE_M is RBE at low doses and low dose rates obtained by extrapolation of data on dose-response for neutrons and the reference radiation. Life-shortening in these studies was due mainly to induction of cancers. When two sets of values are given, they represent alternative interpretations that are consistent with the data.

[b]LL and UL are the lower and upper 68% confidence limits, respectively, corresponding to one standard error.

[c]Results from a study using *X* rays as the reference radiation are omitted.

Table 4. Values of RBE_H and RBE_M of fission neutrons for induction of specific cancers in various strains of mice derived from analysis of selected studies by Edwards (1999)[a]

		RBE_H			RBE_M		
Strain	Cancer	LL[b]	Mean	UL[b]	LL[b]	Mean	UL[b]
RF/Un	Myeloid leukemia	1.7	2.8	4.7	9	19	38
	Lymphoma	2.2	2.9	3.7	2.7	4.7	5.6
RFM							
Male	Myeloid leukemia	2.2	2.8	3.8	—	—	—
Female	Thymic leukemia	3.3	4.1	5.1	12	29	64
	Harderian gland tumor	7	9	11	22	33	47
	Pituitary tumor	5	7	10	17	120	∞
BALB/c							
Female	Lung adenocarcinoma	5.5	7.5	10	12	20	30
	Mammary tumor	2.5 6	3.5 11	5 20	18	27	41
CBA/H	Myeloid leukemia	4	7	10	14	21	36
SAS/4							
Male	Lung adenocarcinoma	3	5	9	—	—	—
Female	Lung adenocarcinoma	5	8	14	—	—	—

[a]See Table 4 of Edwards (1999). RBE_H is RBE at high acute doses of reference high-energy gamma radiation, and RBE_M is RBE at low doses and low dose rates obtained by extrapolation of data on dose-response for neutrons and the reference radiation. When two sets of values are given, they represent alternative interpretations that are consistent with the data. Analysis was based on data in Upton et al. (1970), Ullrich et al. (1976), Ullrich and Preston (1987), Ullrich et al. (1979), Ullrich (1980), Ullrich et al. (1977), Ullrich (1984), Mole and Davids (1982), Mole et al. (1983), and Coggle (1988).

[b]LL and UL are the lower and upper 68% confidence limits, respectively, corresponding to one standard error.

Table 5. Values of RBE_H and RBE_M of fission neutrons for tumor induction in B6CF1 mice derived from analysis of selected study by Edwards (1999)[a]

Tumor	Times of death (days after irradiation)	Sex	RBE_H[b]	RBE_M[b]
Lymphocytic	600-799	Male		6.6 ± 1.8
			2.0 ± 0.3	20 ± 5
			5.7 ± 0.9	12 ± 4
				36 ± 13
		Female	5.4 ± 0.6	8.4 ± 0.7
			11.4 ± 0.6	17.8 ± 1.5
	800-999	Male	2.5 ± 0.5	9.7 ± 1.5
			6.5 ± 1.1	25.2 ± 3.2
		Female	8.5 ± 3.0	8.5 ± 1.8
			17 ± 5	15.8 ± 2.6
Vascular tissue	600-799	Male	4.7 ± 0.6	13.9 ± 2.6
		Female	3.7 ± 1.0	7.2 ± 3.2
	800-999	Male	4.8 ± 1.0	13.7 ± 1.6
		Female	6.4 ± 1.4	8.9 ± 2.0
All epithelial tissue or ovary	600-799	Male	5.5 ± 1.0	23 ± 5
			11.0 ± 1.5	45 ± 7
		Female	10.5 ± 1.5	23 ± 4
	800-999	Male	6.2 ± 1.3	14.4 ± 2.9
			13.4 ± 2.2	31 ± 5
		Female	9.7 ± 1.9	19 ± 6

[a]See Table 5 of Edwards (1999). RBE_H is RBE at high acute doses of reference high-energy gamma radiation, and RBE_M is RBE at low doses and low dose rates obtained by extrapolation of data on dose-response for neutrons and the reference radiation. Analysis was based on data in Grahn et al. (1992). When two sets of values are given, they represent alternative interpretations that are consistent with the data.

[b]Uncertainties are one standard error.

Table 6. Quality factors for neutrons currently used by U.S. Nuclear Regulatory Commission and U.S. Department of Energy[a]

Neutron energy (MeV)	Mean quality factor[b]
≤0.001	2
0.01	2.5
0.1	7.5
0.5	11
1	11
2.5	9
5	8
7	7
10	6.5
14	7.5
20	8
40	7
60	5.5
100	4
≥200	3.5

[a]Values given in NRC (1991) and DOE (1993) are based on calculations and recommendations in NCRP (1971).

[b]Maximum calculated quality factors in a 30-cm diameter sphere of tissue-equivalent material.

Table 7. Values of RBE_M for alpha particles obtained from reviews and analyses of selected studies by the NCRP (1990) and Muirhead et al. (1993)[a]

Endpoint	RBE_M	Reference
Lung tumors (various species)	30 (6-40)	ICRP (1980)[b]
Bone tumors (dogs)	26	NCRP (1990)[c]
Bone tumors (mice)	25	NCRP (1990)[c]
Lung tumors (dogs)	30-60	NCRP (1990)[d]
Bone tumors (dogs)	4-6	Griffith et al. (1991)
Lung tumors (rats)	25	Hahn et al. (1991)
Lung tumors (dogs)	36	Hahn et al. (1991)[e]
Cell transformation (C3H10T½ mouse cells)	10-25	Brenner (1990)
Cell mutation (Chinese hamster cells, V79)	Up to 18	Thacker et al. (1979)
Chromosome aberrations (liver cells of Chinese hamster)	15-20	Brooks et al. (1972); Brooks (1975)
Chromosome aberrations (human lymphocytes)	5-35	Edwards et al. (1980); Purrott et al. (1980)
Germ cell mutations (chromosome fragments, chromosome translocations, dominant lethals)	22-24	Searle et al. (1976)

[a]Based on data presented in Section 7 of NCRP (1990) and Table 7.3 of Muirhead et al. (1993). RBE_M is RBE at low doses and low dose rates of reference radiation obtained by extrapolation of data on dose-response for alpha particles and reference radiation at high doses. Reference radiation in all studies was either beta particles from decay of radionuclides or high-energy gamma rays from ^{60}Co decay.

[b]Range based on analyses of dose-response at 10% and 40% lung tumor incidence for inhalation of soluble and insoluble alpha-emitting radionuclides combined; estimates based on analyses for inhalation of insoluble ^{239}Pu oxide only range from about 10 to nearly 100.

[c]Value based on re-analysis of preliminary data in Mays and Finkel (1980).

[d]Range based on preliminary results from Boecker et al. (1988) and Griffith et al. (1987); value toward upper end of range is not supported by subsequent analysis by Hahn et al. (1991), and value from Boecker et al. (1988) could be as low as 10.

[e]Result based on subsequent analysis of data in Boecker et al. (1988) and Griffith et al. (1987).

Table 8. Dose-response relationships of *X* rays and reference gamma rays for induction of dicentric chromosomes in human lymphocytes[a]

Reference	Radiation	Dose range[b] (Gy)	$\alpha \pm SE$[c] ($\times 10^{-2}$ Gy^{-1})	$\beta \pm SE$[c] ($\times 10^{-2}$ Gy^{-2})
Bauchinger (1984)	220 kVp *X* rays	0.5-4	4.0 ± 0.3	5.98 ± 0.17
	^{60}Co γ rays	0.5-4	1.1 ± 0.4	5.55 ± 0.28
Fabry et al. (1985)	250 kVp *X* rays	0.05-2	4.4 ± 1.0	6.0 ± 1.1
	^{60}Co γ rays	0.05-2	3.0 ± 0.8	4.3 ± 1.0
Lloyd et al. (1986)	250 kVp *X* rays	0.05-6	3.6 ± 0.5	6.67 ± 0.22
	^{60}Co γ rays	0.05-6	1.4 ± 0.4	7.59 ± 0.27
Littlefield et al. (1989)	220 kVp *X* rays	0.25-3.75	4.3 ± 0.8	6.6 ± 0.4
	^{60}Co γ rays	0.25-4	1.6 ± 0.7	5.7 ± 0.3
Brewen and Luippold (1971)[d]	250 kVp *X* rays	0.5-4	9.1 ± 0.2	6.0 ± 0.7
Brewen et al. (1972)[d]	^{60}Co γ rays	0.5-4	3.9 ± 1.1	8.2 ± 0.4
Lloyd et al. (1975)	250 kVp *X* rays	0.05-8	4.8 ± 0.5	6.2 ± 0.3
	^{60}Co γ rays	0.25-8	1.6 ± 0.3	5.0 ± 0.2

[a]See Tables 2.6 and 2.7 of NCRP (1990).

[b]Doses were delivered acutely or over a time period of about 10 minutes or less.

[c]α and β are coefficients of linear and quadratic terms in dose-response relationship, and SE is the standard error.

[d]Results for *X* rays and gamma rays were reported separately.

Table 9. Estimates of RBE_M for *X* rays for induction of dicentric chromosomes in human lymphocytes[a]

Reference	*X* rays	RBE_M (68% CI)[b]
Bauchinger (1984)	220 kVp	3.8 (2.5, 6.5)
Fabry et al. (1985)	250 kVp	1.5 (1.0, 2.2)
Lloyd et al. (1986)	250 kVp	2.6 (1.8, 3.8)
Littlefield et al. (1989)	220 kVp	2.8 (1.7, 5.1)
Brewen and Luippold (1971); Brewen et al. (1972)	250 kVp	2.3 (1.8, 3.3)
Lloyd et al. (1975)	250 kVp	3.0 (2.4, 3.8)

[a]RBE_M is RBE at low doses obtained by extrapolation of linear-quadratic dose-response relationships for *X* rays and reference ^{60}Co gamma rays.

[b]First entry is point estimate calculated by NCRP (1990) as α_X/α_γ, where α_X and α_γ are central estimates of coefficient of linear term in dose-response relationship for *X* rays and gamma rays, respectively, given in Table 8. Second entry in parentheses is 68% confidence interval based on standard errors in α coefficients given in Table 8 and calculated as described in text.